THE PATHOLOGY OF THE KIDNEY.

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Under normal conditions the function of the Kidneys may be classed under two heads, first, a true secretary function, and second, a function which is nearly allide to simple filtration.

The true secreting work of the kidney is performed by the glandular epithelium which lines the convoluted portions of the tubules; various organic substances - urea, uric acid, and a certain proportion of the solids of the urine - are separated from the blood by the secreting cells of these tubules; while the filtration or diffusion of fluid appears to be performed by the tufts of capillaries within the Malpighian capsules; without a doubt the capillaries within the glomeruli are submitted to a high blood pressure, and reference has often been made to the smallness of the efferent vessels as an indication of one possible cause of the high pressure within the Malpighian tuft. This explanation however, scarcely seems conclusive; the efferent vessel is certainly smaller than the afferent but this possibly may be the work of the Malpighian tuft rather than its cause. Undoubtedly a small efferent vessel, if its walls were rigid would give rise to a high blood pressure within the Malpighian tuft, but it appears to be equally probable that the efferent vessel is small as the result of the filtration from the Malpighian capillaries. The efferent vessel has less fluid to carry than the afferent and therefore it is not necessary

that it should be so large. In all probability some salts pass through the Malpighian tuft together with water, as for example the inorganic crystalline salts which doubtless readily diffuse through the membrane.

The presence of a layer of flattened epithelium over the capillary tuft has been said to prevent to a large extent the albuminous constituents of the blood from passing away with the water, and this contention has derived some support from experiment. If the renal arteries are ligatured for a short time, this epithelial coating becomes injured, and the urine is found to be albuminous.

The twofold sources of the constituents of the urine have long been recognised, and numerous experiments tend to show the correctness of this theory. Undoubtedly when the epithelium of the convoluted tubes becomes injured by disease, the excretory power of the kidney is impaired and the interference in function is found to affect the organic substances rather than the water and inorganic salts. In other words, in chronic kidney disease, while the amount of urea and uric acid is largely reduced, the amount of urine passed is frequently slightly in excess of the normal quantity. The diminution of the organic constituents of the urine is the natural result of the alteration of the secreting epithelial

cells, but the alteration in the quantity of the urine is not so easily explained. When the vessels of the kidney are engorged, either as part of an acute inflammatory process, or in consequence of interference with the circulation, owing to chronic disease of the heart or the lungs, the amount of urine is commonly much reduced. With less blood passing through the renal capillaries in a given time less fluid is withdrawn from them. When, on the other hand, the pressure within the renal vessels is increased, as for instance from hypertrophy of the arterioles in chronic kidney disease, an increased filtration is favoured, and the daily amount of fluid excreted is commonly greater than in the healthy state.

These clinical deductions, in support of the two-fold nature of the work of the Malpighian capillaries, and of the convoluted tubes, are in close accordance with the results of experimental investigations upon the healthy kidney. It has been stated for example, from the experimental side that Ribbert has succeeded in extirpating the medullary substance of the kidney while leaving the cortical part intact, and that when he collected the urine which passed through the Malpighian bodies only, before reaching the looped tubules, he found that it was far more watery than that which was secreted by the entire kidney.

The theory receives more satisfactory confirmation from the changes in the urine associated with the relative insignificance of the tubules of the kidney. Hufner has observed that among fishes, frogs, tortoises birds and mammals, the tubes are long or short according as the animal requires water to be re-absorbed in each case.

Nussbaum experimented upon frogs and newts to shew that urea was excreted by the epithelial cells, and he found that sugar and peptone injected into the circulation pass into the urine of a kidney when intact, but not when the renal artery is tied. On the other hand he found that urea was excreted only when the circulation among the tubules was intact, and he further asserted that water was excreted in two ways by the kidney, both by the glomeruli and by the epithelial cells. With regard to the influence of nerves on the secretion of the Kidney, it may be stated that dilatation of the branches of the renal arteries produced through the influence of the vaso motor nerves will cause increase of pressure within the glomeruli, and will result in the passage of an increased quantity of urine. When the pressure is increased by other conditions - such as for instance by an increase in the force, or in the frequency of the heart's beat, by an increase in the volume of the blood, or by a constriction of

of small vessels independently of those of the Kidney the result is seen again in an increased quantity of water. On the other hand whenever the pressure within the glomeruli is diminished it is associated with a diminution in the amount of urine excreted. While, therefore, both clinically and experimentally, it may be regarded as certain that the nitrogenous elements are separated by the epithelium in the convoluted tubes, and that the water and inorganic salts come from the Malpighian tufts, it must be admitted that two points yet remain doubtful; first, whether any fluid passes into the convoluted tubes through the epithelial cells in company with the nitrogenous elements, and secondly, whether, as has been asserted, there is a normal outflow of albumin from the Malpighian tufts, with re-absorption of the albumin by the cells in the tubules.

This paper will be devoted to a discussion of the pathological changes produced in the Kidney by variations in the above mentioned normal processes brought about by certain diseased conditions.
Of these, the most common and most important, clinically, will be considered first, viz. the disease known as Nephritis.
Sir George Johnson contends that in Nephritis (Bright's disease) the true starting point of the condition

is not the Kidney, that in fact it is not a local malady at all but a disease of constitutional origin; and that the proximate cause of the renal disease is in all probability a morbid condition of the blood. Numerous names have been used for this disease. It has been termed: Acute tubal Nephritis, Croupous Nephritis, Acute desquamative Nephritis, Acute Parenchymatous Nephritis, Acute infective Nephritis, and acute Bright's disease. All of these terms are unsatisfactory, many of them fixing the attention on a portion only of the Pathological change, It is preferable therefore to use the term Nephritis.

Acute Nephritis.

Etiology. The influence of age is somewhat marked in connection with acute inflammation of the Kidneys. It occurs mostly in young adults, though it is occasionally seen in early middle age. It is more frequent amongst males than females, and this is probably to be explained by the former being more liable to exposure to the various conditions under which the disease arises. It is also more common amongst the middle and laboring classes, owing to the general circumstances of their occupations, their clothing and their habits.

Amongst young adults cold is one of the most common causes of acute Nephritis, and it is the more likely

to produce this disease when the individual has been exhausted with work or exercise or when the skin has been actively perspiring. The sudden contraction of the cutaneous vessels under these circumstances is likely to be followed by internal engorgement, which starts the subsequent changes in the Kidney. It has also been frequently produced by cold and wet, affecting those who are heated by hunting or by cycling, while exposure after dissipation has also resulted in this disease.

Amongst children it is relatively rare to find acute Nephritis attributable to cold, unless the individual has previously suffered from one of the exanthemata, notably scarlet fever, also small-pox, measles, erysipelas, typhoid, and pneumonia.

The frequent connection of these diseases with renal changes led to the employment of the term "Acute infective Nephritis" under the idea that the Pathological condition was the result of an inflammation excited by Bacteria. It is now known that micro-organisms are absent in most cases of Nephritis, and it is generally held that the renal changes are due to the action of toxic material resulting from the growth of the specific organisms within the body.

Numerous drugs may cause Acute Nephritis, especially those which are absorbed into the blood and eliminated by the Kidney. Prominent among these may be

reckoned cantharides, turpentine, copaiba, phosphorus and alcohol. The condition produced by these substances is probably limited to a temporary congestion leading to albuminuria or haematuria rather than the production of a true Nephritis.

The Pathology of any form of Nephritis may be most conveniently studied if we remember that the different structures of which the Kidney is composed may frequently be affected independently and that when different structures are concurrently affected, the extent to which each part is involved is liable to great variations; Thus the tubules and their epithelium which together constitute the parenchyma of the Kidney, sometimes shew the most marked alterations, and this is especially seen in most of the acute forms of Kidney disease. In more chronic conditions the interstitial tissue, which in the healthy Kidney is only present in small quantity, may increase considerably as the result of inflammatory processes; and thirdly the blood vessels and the glomeruli appear liable to separate affection, and indeed the epithelial cells which coat the capillaries of the glomerules have been described as undergoing an inflammatory process which for some authors, constitutes the primary and perhaps the most important change in the Kidney when diseased.

IN ACUTE NEPHRITIS. The primary changes are undoubtedly connected with the tubules and the glomeruli, and these changes are found to be fairly constant when the Kidney is examined microscopically, although the size, shape, and the general appearance of the Kidney are largely dependent upon the stage the disease has reached when death occurs.

Sometimes the Kidney is very little larger than normal, the cortex is of a dark color and the glomeruli stand out as minute pale spots. Sometime the Kidney is increased considerably in size. It may indeed be swollen to almost twice its normal bulk. It then has a more rounded appearance than usual: the capsule can be separated easily and the surface is pale.

On Section: The increase in size is found to be due mainly to an increase in the thickness of the cortex, which is of a greyish red color, while the central portion, the pyramids, are of a deeper red owing to the congestion of their vessels. Frequently both on the surface and on section bright red spots are found scattered through the Kidney. Sometimes these are due to minute Haemorrhages, sometimes to engorgement of the glomeruli.

In a third variety of Acute Nephritis the degree of congestion is very much greater, so that the color is dark red or brown, and on section the Kidneys have been described as "dripping with blood".

Microscopically. changes are found both within the tubes, in the interstitial tissue, and within the glomeruli although the extent to which each anatomical division is affect, varies considerably in different cases.

The change within the tubes mostly consists in a peculiar opaque appearance of the epithelial cells, these may be swollen and granular or they may be filled with fat granules and they frequently tend to separate readily from the wall of the tubule. The lumen of the tubule is often obliterated by debris of altered epithelial cells, leucocytes, and coagulated albuminous fluid; and sometimes the tubules appear to be blocked with a compact mass of blood corpuscles. Hyaline, epithelial, and blood casts are often seen within the tubes, both in the cortex and in the looped tubules of the pyramids. When the Kidneys are large it is frequently found that leucocytes have exuded from the blood vessels, and that they have been accompanied by an inflammatory fluid; the inflammatory exudations are most marked in the neighborhood of the Malpighian corpuscles. The glomerular changes, upon which some authors have so much insisted, have been described as a form of capsulitis. The capsule is distended by a finely granular mass, enclosing numerous small nuclei, and sometimes haemorrhages occur within the capsule. From both of these conditions it is reasonable to assume

that the glomerular tuft has been subjected to considerable pressure, which must necessarily have interfered with the circulation through the capillaries, as well as with the excretion of water. Klebbs, Greenfield, and Klein, consider that the nuclei of the capillary tufts in the glomerulus proliferate, and that this proliferation is accompanied by an excessive growth of nuclei within the capsule, which leads to adhesion between it and the glomerulus, and ultimately to compression and atrophy of the latter. This condition, in which a few, or a very large proportion of glomeruli may be affected, has been termed "glomerulo Nephritis" and it appears to be most common in the form of acute Nephritis consecutive to Scarlet Fever. In such cases the lesions within the tubules have been considered to be secondary to the abolition of the work of the glomerulus.

Scarletinal Nephritis. The frequency with which acute Nephritis accompanies and compliactes cases of Scarlet Fever appears to separate this type from other forms of Acute Nephritis, and its pathology being somewhat different, it merits separate consideration. From the frequency of its occurrence there can be little doubt that it depends upon the action of some poison peculiar to Scarlet Fever. Doctor Hillier and Dr Dickinson found albuminuria in

half the cases of Scarletina they investigated, and it appears probable that although in some cases the Albuminuria should be regarded as pyrexial, yet in a large number the albuminuria was distinctly due to renal change.

Pathology. Friedländer describes three distinct forms of changes in the Kidney associated with Scarlet Fever.

1. Initial catarrhal, the usual form.
2. The large limp haemorrhagic Kidney, interstitial septic form.
3. Nephritis post-scarletinosa, the glomerular form.

The first appears with the onset of the Fever, and usually clears up after one or two weeks. The second form appears early but is very severe, and is also usually connected with so called diptheritic affections, cervical phlegmon etc; the third is an affection characteristic of scarlet fever, and is often manifested by oedema, hypertrophied heart, suppression of the urine, and death. This form constitutes scarletinal Nephritis - the disease now under consideration.

The appearances of the Kidney are largely dependent upon the duration of the disease, and for descriptive purposes it will be convenient to separate three broadly differentiated types of scarletinal Nephritis,

but the boundaries of this arrangement are ill-defined, and the separate types frequently merge.

When death has occurred during the first or second week, the kidneys are very little altered in size. They appear engorged and the Capsule can be readily separated. The surface may shew radiating red lines of injection or small extravasations. On section, the whole of the parenchyma may be of a chocolate hue from intense engorgement.

From the second to the sixth week the Kidneys are enlarged and firm, and the surface is more pale and mottled: yellowish spots are scattered over the surface of the Kidney, which is either of normal color or more pale than usual. These appearances are seen to extend through the cortical layer, but the medullary substance is still hyperaemic, from its junction with the cortex almost to the ends of the papillae. The hyperaemia is often associated with striae of a deep color, passing along the pyramids. At a later stage contraction occurs so that the Kidneys may be of normal size, or even somewhat smaller. They may still be pale in color, but they are much more firm, and they may even be rather difficult to cut.

Microscopically it is seen that scarletinal Nephritis mainly affects the glomeruli and the tissues in their neighborhood. In the early stages the vessels of

these parts are found to be crowded with masses of corpuscles, which are most numerous between the vessels of the tuft of capillaries, while they are also frequently found in considerable numbers outside the tuft but within Bowmans Capsule. The vessels of the tuft sometimes rupture, and Bowman's capsule becomes filled with blood corpuscles, which are often seen extending down the tubules: compact masses of corpuscles due to haemorrhage may also be found in lines between the tubules. The epithelial cells in the convoluted tubes are enlarged and cloudy, and they may be swollen to such an extent as to almost obliterate the lumen. Similar changes affect the epithelial cells lining Bowman's capsule.

At a later stage, the round cells in the neighborhood of the vessels and round the capsules of the glomeruli proliferate, and the epithelial cells lining Bowman's capsule undergo rapid desquamation: hence the space between the tuft and the capsule is filled with collections of exfoliated epithelium. The appearance of thecapillary tufts depends upon the extent of the changes within the capsule: the tufts may be engorged so that they occupy the whole of the space within the capsule, or they may be compressed and relatively empty, owing to the pressure exerted upon them by the desquamated epithelium and the haemorrhagic effusions.

The epithelial cells in the convoluted tubes are more granular and opaque at this stage, and undergo rapid desquamation; hence the tubes are often found choked with granular debris which may be entangled in the form of casts.

When the Kidney is undergoing contraction in the third stage, Bowman's capsule is greatly thickened, and the whole Malpighian body is larger than usual, owing to the great increase of nucleated cells surrounding and compressing the capillary tuft. This solidification of the glomerular tuft may therefore be due to a combined growth of epithelial cells outside the capillary membrane, and of other cells, connective tissue corpuscles, and leucocytes within it. Similar increase of connective tissue corpuscles is found round the outside of Bowman's Capsule, and between the convoluted tubes which may be compressed and distorted by the new growth. The epithelial cells of the convoluted tubes are generally flattened at this stage, and the lumen, which may be larger than in the normal condition is often filled with debris and casts.

In all these stages the interlobular arteries and arterioles are thickened and prominent. The extent to which they are affected increasing with the duration of the disease.

In view of the well marked nature of the alterations

within and around the capsule in scarletinal Nephritis, both Langhans and Nauwerk have suggested that vascular alterations, limited probably to the glomeruli, might cause fatal uraemia, and hence they have described this condition as glomerular Nephritis, and they consider the changes in the tubules and in the interstitial to be the result of the glomerulitis. This view has been vigorously contested, and, while it must be admitted that the alterations in the tubules and in the glomeruli nearly always co-exist, there is little doubt that the glomerular changes are not invariably the first to appear.

Chronic nephritis.

This form of nephritis has been described under numerous names which are intended to indicate the naked eye appearances of the Kidney, or to summarise the microscopic pathological changes. Large white Kidney, non-desquamative nephritis, chronic parenchymatous nephritis, chronic tubal nephritis, chronic catarrhal nephritis, chronic diffuse nephritis; nearly all these terms are open to objections, the restrictive terms are scarcely applicable to any single case, and although changes occur within the tubes and affect the parenchyma, the degree with which each

tissue is affected will vary considerably. In the early stages the condition here described as chronic nephritis undoubtedly corresponds with the pale marbled, or mottled Kidney, and with the large smooth granular Kidney described by Bright; but in the later stages the pathological appearances are widely different from this type.

Etiology. Chronic Nephritis in the large majority of cases is the result of a continuance of the changes due to acute nephritis. It therefore occurs later in life. It is most common between the ages of 25 and 40, and in this respect differs essentially from the age affected by Cirrhosis of the Kidney, (chronic interstitial nephritis) a disease occurring more commonly in persons about 50. Many cases of chronic nephritis result from acute scarletinal nephritis. The influence of cold in the production of acute nephritis has already been mentioned and there is common agreement that frequent exposure to wet and residence in damp and cold climates also favor the onset of the chronic form. Chronic supuration resulting from phthisis, necrosed bone, or other cause may produce chronic affection of the Kidneys. The change that results is usually a form of lardaceous disease, mainly affecting the Malpighian tufts; but this is very frequently accompanied by chronic nephritis; in fact

it is relatively rare to find lardaceous disease in the Kidney without some form of chronic nephritis. Malaria, Syphilis, lead, and alcohol, also cause chronic disease of the Kidney.

Although it is commonly considered that chronic nephritis has its origin in an acute attack, and although this position is not materially shaken by the statement that it may sometimes commence as a slow lingering process yet, it is interesting to note that the changes are not universally attributed to anatomical lesions. According to one theory the disease depends upon an alteration of the blood, and the renal lesion plays a secondary part.

Another hypothesis is that it is part of a general nutrative disturbance which interferes with the assimilation of albumen, and the source of this interference has been attributed to some alteration of the functions of the skin, whether these functions are disturbed by cold, by interference with the freedom of perspiration, or by some form of skin disease. It must be admitted that in many forms of chronic Kidney disease the skin does not act with its customary freedom, and Semmola has compared persons with chronic nephritis to animals whose skin has been varnished.

Another interesting theory is that chronic renal disease results from irritation by various toxic substances produced by incomplete oxidation of the

tissues, as, for example creatin, creatinin, leucin, tyrosin, and xanthin. This theory may to a certain extent receive support from the undoubted occurrence of chronic nephritis in connection with diabetes. Other observers attribute chronic nephritis to some disease of the central nervous system by means of which an alteration is produced in the flow of blood to the Kidneys.

Pathology. The appearances of the Kidney in chronic nephritis are subject to the greatest variations, for which one is wholly unprepared in view of the great similarity of symptoms. Thus, cases which are practically indistinguishable so far as the symptoms are concerned, may be associated with either large white Kidney, or with the smaller granular Kidney, or with the speckled Kidney. Sometimes, too, the naked eye appearances indicate very little, if any, deviation from the normal type. In spite of these differences however, the microscopic examination will generally indicate fundamental points of resemblance.

If the disease terminates within a year of the first development of symptoms, the kidney is as a rule found to be larger than usual. Sometimes this enlargement is extreme, and the kidneys may be from twice to three times their normal size. Fagge mentions three cases in which the weight of a pair of kidneys

was 28½ to 29 ounces.

The capsule is usually readily detached, and the surface of the Kidney is smooth and somewhat pale; sometimes the Capsule cannot be so easily stripped off, and after removal the surface is finely granular and perhaps somewhat torn. The cortical part of the kidney usually shows stellate haemorrhages or engorgements of the stellate veins.

On section whitish or yellowish streaks and spots may be found passing through the substance of the cortex, and these may give the appearance of the kidney having been strewn with fine sand. The cortex is considerably thickened so much so that it may nearly equal the thickness of the cones.

The normal relation between the cortex and the medulla is generally as one to three; in chronic nephritis the cortex is so much increased that it may be from one to two or the parts may even be of equal thickness. The cones are usually of a darker red than the cortex, and form a marked contrast with the pale cortical portion of the Kidney.

Microscopically it is seen that, as in acute nephritis, the changes are not uniformly distributed through the kidney, but that they affect certain areas while intervening tracts of tissue appear to be normal. All the structures of the kidney may, however, be involved in chronic nephritis. Alterations are found

in the tubules, the Malpighian tufts, the interstitial tissues and in the blood vessels. In the convoluted tubules the epithelium is generally swollen, cloudy and less transparent than usual; the degree of swelling may be sufficient almost to occlude the tubule, and also to interfere with the freedom of circulation. The combined result of the distension of the tubes and of the compression of the blood vessels, is seen in the white or grey color of the cortex. Sometimes, however, the tubules are found with the epithilium desquamated or proliferating; sometimes the tubules may be entirely denuded of epithelium. Occasionally the renal epithelium is replaced by thin, flattened cells, the lumen of the tube being considerably increased in diameter.

The extent to which the interstitial tissue is affected will also vary considerably. It is generally considered that there is cullular infiltration of the spaces between the tubules, which subsequently results in the formation of fibrous connective tissue, and undergoes contraction. The extent to which this development of interstitial tissue occurs may lead ultimately to irregularities of the surface of the kidney, and may be responsible in some cases for the partial adhesion of the capsule.

In another theory the nuclei of the interstitial inflammation have been referred to the obliterated

bloodvessels, while the tracts of fibrous tissue have been described as tracts in which the tubules have been similarly obliterated. Inasmuch, however, as the large white kidney undoubtedly occurs in a later contracted form, there seems to be every reason to believe that the contraction is due to the subsequent alterations in an overgrowth of fibrous tissue.

The changes affecting the Malpighian tufts are similar to those characteristic of acute nephritis. As the result of subsequent changes however, the glomeruli appear more or less atrophied while the Capsule of Bowman is considerably thickened, and its epithelial lining has undergone degeneration. Frequently the Malpighian tuft is infiltrated with leucocytes, and the surrounding tissue also presents an excess of nuclei.

Greenfield has described three changes connected with the capsule, (1) Pericapsulitis, or cellular infiltration and tissue formation occurring chiefly outside the capsule; (2) Hyaline thickening of the capsule itself; (3) Endo-capsulitis, or the formation of concantric laminae of cells and tissue inside the capsule, between it and the glomerular tuft. In chronic cases these concentric laminae are due to a new formation between the capsule and the tuft, and the capillaries meanwhile undergo hyaline thickening. The capsule in advanced cases is striated, and

concentrically thickened, and blends with the sclerosed glomerular tuft (Auld). These changes are not invariably present. They are most common in chronic nephritis, originating in scarletinal nephritis, but even in such cases they are occasionally absent . The branches of small arteries within the kidney are invariably found to be considerably thickened, the hypertrophy affecting the muscular coat as described by Sir George Johnson. Some thickening however of the fibrous wall of the vessel must also be recognised. The vessels alone may sometimes appear to be engorged with blood, but more often some of the tubes also shew the presence of red blood corpuscles closely packed together, while in other parts of the kidney the lumen of the tubes may be occupied by casts. The increase in size of the kidney is generally due to inflammatory deposits in the parenchyma and interstitial tissue. If, however, the overgrowth of interstitial elements is associated with marked atrophy of the parenchyma, the absolute size of the kidney may not appear to have undergone any very material alteration. On the other hand if the new connective tissue undergoes contraction the size, weight, and appearance of the kidney will differ greatly from the appearances above described. The kidney however remains more pale and mottled than in the normal condition, and though the surface may become uneven and granular, the color

alone indicates that the case is one of contracted kidney, instead of being one of small red granular, or cirrhosed kidney. When the kidney is diminished in size in this way, the chief change is one affecting the cortex, which becomes narrower than before, and on microscopic examination of the cortex, wide tracts of fibrous tissue almost homogeneous in appearance may be found lying either immediately below the capsule or in the course of the inter-lobular arteries. In this tissue, atrophied tubes, Malpighian bodies, and blood vessels may be found, and in the intervening spaces between these patches of fibrous tissue, the epithelial cells of the convoluted tubes are seen to be fatty and opaque, and small cysts, similar to those so common in renal cirrhosis, often result from blocking of the tubes.

In other respects the changes of the Malpighian bodies, of Bowman's capsule, and of the smaller arteries are similar to the changes described in connection with the large white kidney.

From the above description it will be seen that although the macroscopic appearance of the kidney are so very dissimilar, yet the microscopic changes are merely those which might be anticipated as the result of a chronic process. In fact, the difference in appearance depends largely upon the length of time that has elapsed between the commencement of the

disease and its termination. Neither condition is essentially parenchymatous or essentially interstitial. The parenchymatous changes may be more apparent in the earlier stages of the large white kidney, while the interstitial changes predominate in the later conditions, but all the tissues are simultaneously affected, and the size of the kidney seems undoubtedly to depend upon the degree of contraction and atrophy that has succeeded the inflammatory process. Lardaceous changes may occasionally develope in connection with chronic nephritis, but they are of slight extent, and do not materially affect the size of the kidney.

The tendency of chronic nephritis is undoubtedly towards atrophy and contraction, and indeed some authors consider that the later stages, when the kidney is small, pale, with a narrow cortex and granular surface have always been preceded by an earlier stage in which it was enlarged, white, and with a thickened cortex.

Chronic Interstitial Nephritis - Cirrhosis of the kidney.

This condition has also been termed: red granular kidney, granular atrophy, granular degeneration, gouty kidney, chronic gouty nephritis, chronic Bright's disease and renal cirrhosis.

The course of this disease is essentially slow and chronic from its commencement. It mainly occurs towards the later part of middle life and during advanced age. As with the contracted kidney of chronic nephritis previously described, the part mainly affected is the renal cortex, which is greatly diminished in thickness, while the surface of the kidney becomes uneven and granular. During a large part of the course of the disease the urine is copious and of low specific gravity. The amount of albumin passed is very small. Indeed, in many cases there may be no albumin for days or weeks at a time. Dropsy is mostly absent with this form of kidney disease, unless it is complicated by acute or chronic neprhitis; but on the other hand the changes in the heart and arteries are far more developed than in other renal affections.

Etiology. In a disease which is so chronic in its nature, the etiology necessarily becomes somewhat uncertain. However, there seems to be good ground for believing that the supervention of cirrhosis of the kidney may be influenced by the age and sex of the individual as well by heredity,

It is rare amongst children or even amongst young adults. Below the age of 30 it is extremely rare to find typical examples of cirrhotic kidney, the majority of cases occurring between the ages of 40 and 60. With regard to sex, it occurs far more frequently among males than females, this is no doubt the result of the grater exposure of the male sex to the conditions likely to give rise to the disease.

The intimate connection of renal cirrhosis with gout has long been a matter of common observation. It is generally held that gout predisposes to the development of renal cirrhosis. It has also been held that in many cases gout is the consequence rather than the cause of the renal affection, and that it results from the failure of the kidney to perform its normal eliminative function. It is far more probable that renal cirrhosis follows from the constant irritation due to the deposition of uric acid in the solid form within the substance of the kidney.

Like Gout, dyspepsia has been regarded both as the cause and the result of renal cirrhosis. The late Sir George Johnson maintained that renal degeneration is brought about by the long continued elimination by the kidneys of the products of faulty digestion; and on the other hand it has been argued that dyspepsia may be looked upon as a symptom of the kidney

condition.

Other factors in the causation of renal cirrhosis are eating and drinking to excess, lead poisoning, damp climates and rapid alterations of temperature.

Pathology. The most striking change of the morbid anatomy of cirrhosis of the kidney in advanced form consists in the size of the kidneys. These are usually very much diminished in size but the diminution rarely affects both kidneys to the same extent. The combined weight of the two kidneys may be even less than the weight of a single normal kidney. Corresponding with the diminution in size, an increase in the amount of fat enveloping the kidney is usually to be noticed.

In the early stages however, there may be very little alteration in the size or color of the kidneys, and the most prominent characteristic may be the increase in the thickness of the capsule and the greater difficulty of its detachment from the surface of the kidney.

Generally the surface of the kidney is roughened and granular, and of a deep reddish brown color; the capsule can only be detached with difficulty, and portions of the kidney may perhaps tear away with the capsule as it is stripped off. Sometimes the roughened granular surface is covered with little

yellow spots, and frequently small cysts may be found dotted about near the surface of the kidney, and occasionally small white nodules may be found similarly dotted about. The kidney as a whole is firm and resistent, being at times rather difficult to cut, owing to the increased amount of fibrous tissue contained in the organ. On section - it is at once seen that the diminution in the size of the kidney is mainly due to the diminution in the thickness of the cortex. The cysts and white nodules above mentioned are more evident in the cut surface, and in addition, cicatricial tissue may often be observed spreading inwards from the cortex towards the medulla. These cysts are commonly regarded as resulting from the occlusion of the renal tubules near the glomeruli, with consequent distention of Bowman's capsule. The Malpighian bodies are sometimes reduced in size as well as in number, and are frequently found immediately below the capsule of the kidney, instead of being separated from it by a short interval. The pyramidal portions of the kidney are generally of a dark color, and white lines sometimes occupy the position of the straight tubes of the medulla, and indicate the presence of crystalline deposits of urates.

Microscopically the development of fibrous tissue is seen to be extremely irregular: portions of gland

structure, apparently in a healthy condition may intervene: sometimes in the triangular patches of fibrous tissue numerous nuclei are seen: at other times the nuclei are more rare, and the tissue appears more distinctly fibrillated and organised. Compressed and atrophic tubuli uriniferi are occasionally found scattered amongst the new tissue, and if these contain epithelium the cells are usually in a state of fatty degeneration. The connective tissue round the convoluted tubes is very often increased in amount, and the capsules are greatly thickened by proliferation, which tends to encroach upon the space normally occupied by the glomerular tuft, and may ultimately lead to obliteration of the glomerulus.
These atrophic changes in connection with the Malpighian tufts are often regarded as the earliest features of cirrhosis of the kidney, while the atrophy of the tubules and the development of the interstitial changes are regarded as secondary results.
The tubes appear, in some cases to be denuded of their epithelium: in others, to be somewhat distended. Occasionally in the atrophic tissue they may be plugged with colloid material, and distinct casts of considerable diameter may be found obstructing their lumen.
The white nodules are seen to consist of small accumulations of epithelial cells. In the straight tubes of the pyramids the epithelium, as a rule is

normal, but crystals of urate of sodium may be found occasionally within the lumen.

The blood vessels of the kidney are often markedly altered: considerable discussion has arisen as to whether these changes affecting the smallest arteries are secondary to the alterations in the kidney, or whether they are primary changes which precede the alterations in the gland structure. The small arteries may be affected in various ways, but the main change is an increase in the thickness of the walls of the vessel, together with diminution of its calibre. This increase of thickness may be due to an overgrowth of the intima, occurring without material proliferation of nuclei, and may almost entirely occlude the blood vessel. This form of affection corresponds closely with the endarteritis obliterans described by Cornil and Ranvier. Another affection of the small arteries consists in the hyaline fibroid degeneration, which was originally described by the late Sir William Gull, and the late Dr Sutton as arterio capillary fibrosis. In this condition there is some increase in the amount of tissue outside the muscular tissue, and this change was by Gull and Sutton, believed to be the form of alteration of the small arteries most characteristic of contracted kidney. This muscular hypertrophy undoubtedly occurs at a very early stage of renal cirrhosis, and according

to Dr Dickinson it is to be ascribed to some assumed change in the structure of the capillaries rather than as a consequence of cardiac hypertrophy, as originally believed by Sir George Johnson.

Lardaceous disease of the Kidney.

This condition differs from the former ones in the fact that it occurs as a sequel to other well known conditions, and not as a primary renal affection. It presents another distinctive characteristic in being frequently allied with similar changes in other organs, such as the liver and the spleen.

By itself, lardaceous disease of the kidney does not truly represent a form of so called Bright's disease, but the lardaceous change is very frequently accompanied by one or other of the forms of chronic nephritis which have been already mentioned.

The primary change of lardaceous disease is one affecting the renal vessels: the other constituents of the kidney may sometimes be intact - more commonly they shew signs of chronic inflammation.

The disease has been described under numerous different names, as for example, waxy, colloid, amyloid, albuminous, or scrofulous enlargement; the selection of the name being frequently due to ideas respecting the chemical changes produced in the vessels of the kidney. The disease commonly results either from protracted supuration or from syphilis. Tubercular changes are sometimes credited with being able to induce lardaceous disease of the kidney, but it is doubtful whether this is possible unless the tuberculosis is associated with profuse suppuration.

Etiology. Primary lardaceous change in the kidney is very rare. It is usually possible to associate the disease with some other constitutional affection producing marked cachexia. It is said to be most frequent in patients from 20 to 50 years of age, but it has been observed in children who have suffered from chronic suppuration. Bartel assumes that suppuration is insufficient to cause lardaceous change unless the suppurating surface is enabled to come into contact with the atmosphere, and he found that so far as pulmonary tuberculosis was concerned, degeneration of the kidney was more prone to occur after the formation of a cavity in the lung. Chronic disease of bones leading to necrosis and profuse suppuration, such as

disease of the hip joints, and scrofulous caries, are very often associated with this condition. Lardaceous change also occurs with Syphilis, and it is notable that it may occur with confirmed Syphilis, and with hereditary Syphilis, when there has been no ulceration or suppuration, and when the cachetic symptoms have not been well marked.

Other varieties of suppuration predispose to, or induce this disease, e.g. it occurs with empyema, with chronic peritonitis, abscess in the kidney, malarial cachexia and with puerperal and other forms of suppuration connected with the female generative organs. The extent to which other organs are simultaneously affected varies considerably. The small arteries of the liver, when tested with iodine shew the degenerative change similar to those in the kidney. The spleen is commonly affected to a greater extent. Similar alterations have been described in the supra-renal capsule, also in the small arteries of the intestine, and in the vessels of the pancreas and lymphatic glands.

Pathology. The appearances of the kidney depend upon the duration of the disease. In the early stages the kidney may appear normal, the size is unaltered, but the capsule detaches more readily than usual, and the surface is somewhat paler and anaemic. More often the kidney is greatly increased

in size, it may even be as much as twice its normal size. On removal of the capsule, the organ is found to be perfectly smooth and anaemic, and the same appearance of anaemia is found to extend throughout the cortex, the section looking shiny, polished and pale. Spots of a yellowish red color may be seen on its surface and on section of the kidney. Similar spots of a greyish white, semi-translucent appearance are found in the position of the Malpighian bodies. The pyramids are of a dark red color, offering a marked contrast to the yellow pale appearance of the cortex. When the cut surface is washed and treated with an aqueous solution of iodine, the previously translucent spots assume a dark mahogany color. The dark color also appears sometimes in the form of streaks corresponding in position with the vasa recta. When the disease has lasted longer, the appearance of the kidney is very similar to that of the contracted stage of chronic nephritis, though it may be distinguished from the latter by the surface being paler, and by the presence of small sparkling spots. In all probability, both the last mentioned forms owe their size to associated types of chronic nephritis rather than to the lardaceous changes, which in themselves cause little or no alteration in the size of the organ.

Of course, besides these three types described, there

occur transitional stages.

The microscopic appearances differ considerably, according to the stage of the disease, and according to the degree of association with chronich nephritic changes. So far as the purely lardaceous changes are concerned, these affect by preference the glomeruli and the afferent vessels. Sometimes the majority of the glomeruli throughout the kidney shew degenerative changes, sometimes only those in a limited portion are affected. Even in a single Malpighian tuft some of the capillary loops may have escaped, while in other parts the whole tuft appears to be swollen and partially opaque. It was at one time supposed that lardaceous degeneration obliterated the calibre of the blood vessels, but it appears that although the circulation through these vessels may be impaired, it is not arrested, since it is possible in inject the Malpighian loops.

Professor Greenfield states that the various parts of the kidney are involved in the following order:-

(1) Afferent arterioles.

(2) Groups of glomerular capillaries, especially those of the superficial cortex.

(3) The arteriolae rectae.

(4) The efferent arterioles and the capillaries into which they break up.

(5) The capsule of the Malpighian body.

(6) The capillaries which run between the bundles of straight tubes.

(7) The basement membrane of the convoluted tubules.

(8) The large interlobular arteries.

(9) The walls of the straight tubules, especially near the papillae.

(10) The large branches of arteries and veins in the boundary area.

(11) The connective tissue around the collecting tubules at the tips of the papillae.

(12) ~~In rare cases,~~ the epithelial cells.

In considering the various forms of disease of the kidneys, it is proper to consider their influence on the functions of these organs. The pathology of diseases of the kidney may be divided into two series of phenomena: first, the pathological results of diseases of these organs; and secondly, the mode of production of the diseases themselves.
Diseases involving the kidneys tend to produce one or more of the following pathological defects.

(1) Alterations in the composition of the urine.

(2) Oedema.

(3) Uraemia.

(4) Cardio vascular changes.

(5) Marasmus and Anaemia.

(1) Alterations in the urine. The normal flow of urine depends upon the activity of the glomerular epithelium, and on the rate of blood flow through the vessels. The urinary flow is diminished as the result of morbid conditions affecting one or more of the following mechanisms.

(a) Circulatory changes. Firstly - the direct action of various substances on the renal vessels. Substances may, by acting on the renal vessels bring about a diminution in quantity of urine, or even actual suppression by causing vascular constriction.

Frequently this constriction, even if extreme in amount, is followed by dilatation, In many cases depending upon the damage to the vessel wall by the constricting substance, as by turpentine.

This action of substances on the renal vessels is a direct one as shewn by the fact that division of the renal plexus has little effect on the phenomena, and further, that the characteristic effects can be produced in a kidney, excised from the body, through which an artificial circulation is maintained.

Secondly - Indirect, or reflex effects on the renal vessels produced through the nervous system. Constriction of the renal blood vessels produced by reflex excitation is not so likely to lead to diminution or suppression of the urinary flow as direct excitation, since on reflex excitation the local effect is liable to be accompanied by a general constriction, and thus the flow through the kidney is not diminished to the same extent. Stimulation and excitation of the central ends of the lower dorsal nerves produces reflex dilatation of the kidney along with a general constriction.

(b) Epithelial changes. Interference with the renal circulation whether by the produc-

tion of constriction or dilatation, is followed very quickly by changes in the renal epithelium; and these are undoubtedly largely responsible not only for variations in the amount of the urine, but also for alterations in the composition. The direct toxic action of substances on epithelium as in microbic diseases, e.g. diphtheria, commonly produces anuria, and in fatal cases there are no signs of any very profound lesions of the vessels of the kidney. It is probable that in these cases suppression is brought about by the action of the morbid poisons on the epithelial elements. This is in striking contrast to the suppression, of acute nephritis, as of scarlet fever where the changes in the blood vessels are very marked. The increased flow of urine in cirrhosis has been supposed to be dependent on the heightened arterial tension, increasing the rate of flow through the remaining kidney substance. The increased flow cannot very well be due simply to increased blood pressure favoring filtration, inasmuch as, physiologically, the flow of the amount of urine is not dependent upon the absolute blood pressure of the renal vessels, but upon the rate of flow through the renal vessels.

The increase seen in renal cirrhosis is somewhat similar to the increase seen after experimental removal of portions of the kidney, and it may perhaps be dependent rather upon the diminution in the available kidney substance than upon the increased blood pressure. It is possible that the increase in the amount of urine may to a certain extent be an indication of the degree of destruction of the kidney substance. It is certainly remarkable how grat quantities of urine are passed in advanced fibroid changes of the kidney: changes so wide-spread and extensive that but little kidney structure may remain. In amyloid disease, the increased flow is supposed to depend upon the increased permeability of the glomerular tuft.. In chronic nephritis in which the amount of interstitial change is frequently considerable, the flow is also increased, and here the cardio-vascular changes are often by no means so well marked as in cases of so called granular kidney. It is difficult to say whether in these cases the increased flow is dependent simply on the increased blood pressure, or whether, here also it is related to the destruction of kidney substance.

(2) Oedema. - is a frequent accompaniment of renal disease, but it is a variable association. Some diseases of the kidney never cause dropsy, and no disease of the kidney causes it always. Dropsy is peculiarly associated with Bright's disease, acute and chronic, but even in this malady its occurrence is not invariable, and acute nephritis of the severest type may occur without the presence of any dropsy. It is most frequent in the cases of Bright's disease dependent upon scarlet fever, cold, and alcoholism. It is remarkably frequent in large white kidney, not so common in small white kidney. It is also frequent in waxy kidney.

Renal dropsy is associated with diminution in the amount of urine excreted, so that an increase in the dropsy is always associated with a corresponding diminution in the amount of urine voided, and conversely an increased flow of urine is associated with a subsidence in the amount of dropsy.

The dropsy of renal disease affects more especially the subcutaneous tissues: there are also dropsical accumulations in the serous cavities, more especially the pleural cavities; and also, oedema of solid organs occurs. The fluid found in dropsy of renal disease contains a remarkably small percentage of proteid, which is no doubt explainable by the fact that there is a continual loss of albuminous substances owing

to the albuminuria.; it contains large quantities of nitrogenous extractives, more especially in uraemia. The causation of renal dropsy is obscure. From a pathological point of view the dropsical transudations may be looked upon as accumulations of more or less abnormal lymph.- abnormal especially from the presence of a small amount of proteid matter and the large amount of extractives. An increased transudation of lymph must as far as is known, be dependent ultimately either on primary alterations in the wall of the capillaries increasing their permeability, the blood flow through them and the blood pressure in them, remaining normal; or else upon an alteration in the blood pressure and blood flow in the capillaries themselves.

The most plausible explanation of the dropsy in certain forms of renal disease is to assume that the capillary walls have been damaged probably by some material in the blood stream, and that this, together with the hydraemic plethora leads to dropsy. Cohnheim's view, that the dropsy is a kind of subacute inflammation of the skin structures, due to deficient excretory activity of the kidney, is negatived by the composition of the fluid, and by the facts that the dropsy is not limited to the skin, and that complete suppression does not cause dropsy.

(3) Uraemia more or less severe may occur in

almost all diseases of the kidney. It is seen in congestion, in nephritis, in renal cirrhosis, in waxy kidney, in tuberculosis, and calculus disease. Patients may die of uraemia with complete suppression and with but few signs of serious disease of the kidney. It may be sudden in onset and rapid in its course or it may be gradual in onset and slow in its course, in other words, acute or chronic. In type-Uraemia may be classified as nervous, and gastro-intestinal. In the former the symptoms point to disturbance of the nervous system, such as delirium, coma, convulsions: in the latter, to disturbance of the gastro-intestinal functions; nausea, vomiting, diarrhoea,
The nervous symptoms of Uraemia have been explained on the one hand by assuming that they are due to excitation or paralysis of the nerve structures by the changed physical conditions, brought about by cerebral oedema or cerebral anaemia; and on the other hand by supposing them due to the action on the nerve cells on one or more poisons circulating in the blood stream. The majority of observers look upon uraemia as dependent on the presence of toxic material in the blood, and the excitation of the nervous structures by this poison. No such poison has yet been separated and identified, and the great variety of uraemic manifestations has suggested the possibility that more than one toxic body is present.

The toxic substance may appear in the blood under one or more of the following conditions.-

(1) A substance which normally is excreted, is retained.

(2) The abnormal decomposition in the blood or tissues of such a body.

(3) The formation of abnormal products of metabolism by the tissues.

The first is the simplest explanation of Uraemia, and one very generally accepted. In many cases of subacute and chronic uraemia, and in the violent form seen in acute nephritis the quantity of urine excreted is often very small, and examination of the blood shews the presence of greatly increased quantities of nitrogenous extractives. The amount of urea in the blood may be twenty times greater than normal, and although this substance may not be directly answerable for the effects produced, its presence in these large amounts serves as an index to the amount of other and perhaps unknown bodies possessing toxic actions, which may be present in large quantities.

Many observers hold the view that owing to the diminished excretory activity of the kidney, the retained urinary constituents undergo decomposition, either in the blood at large, or in the alimentary canal. It has been suggested that the urea decomposes carbonate of ammonia, and that the toxic phenomena of uraemia

are due to the presence of this body. Carbonate of ammonia when injected into the circulation will undoubtedly produce many symptoms characteristic of uraemia, such as convulsions, and dyspnoea. Many observers however, have failed to detect ammonia in the blood in fatal cases, and for this reason the suggestion has not received any large measure of support.

(4) Cardio vascular changes.- in renal disease are very widespread, especially in renal cirrhosis and chronic nephritis. The pathological changes produced involve the heart and large and small arteries, the former becomes hypertrophied. The large arteries lose their elasticity, and the inner coat becomes atheromatous. The smaller arteries have their coats very much thickened, and this thickening affects mainly the internal coats; the middle coat also shews an increase in the amount of muscular tissue. The arterial changes though frequently widespread are most marked in the vessels of the kidney itself. In addition, miliary aneurysms are common, especially in the cerebral vessels. The cirrhotic kidney and certain forms of chronic Bright's, are the renal lesions most frequently associated with the presence of miliary aneurysms, and hence these are the renal diseases in which cerebral haemorrhage is most prone to occur.

Hyaline changes in the capillaries, e.g. in those of

the glomeruli are common.

These widespread lesions of the vascular system are most extensive in certain cases of renal cirrhosis, more especially in that condition known as "red granular kidney" or "Raspberry kidney", which occurs in middle aged persons: and the greater the arterial disease, the greater the cardiac hypertrophy. The vascular lesions are also fairly well marked in chronic Bright's where there is much fibroid change in the kidney.

The amyloid kidney is not associated with any profound arterial changes except those necessarily associated with the presence of waxy disease in the body, and the heart in these cases is not hypertrophied.

(5) Marasmus and Anaemia. Renal disease frequently produces well marked anaemia and also great wasting. The wasting is due to impaired nutrition dependent on serious disorders of the gastro -intestinal tract. The quantities of albumin lost in urine are often considerable, especially in crhonic Bright's and in this way the nutrition is still further affected. Wasting however, may be a marked feature of renal cirrhosis in which there may be slight gastro-intestinal disturbance, and in which albuminurea is always slight. In these cases the emaciation is probably dependent on an increased disintegration of the proteid tissues, more especially the muscles.

Anaemia is especially associated with chronic Bright's disease in which there is marked dropsy.

Albuminuria - being a constant and important symptom of renal disease, it would be well to briefly discuss its bearing towards the pathology of the kidney.
In Bright's disease, the albuminuria is due to the damage and the shedding of the renal epithelium in the glomeruli and tubules. Even in renal cirrhosis where the albuminuria has been attributed to the high blood pressure, it is more probably due, perhaps, to the accompanying epithelial lesions; for although the main lesion is in the interstitial tissue yet in this disease there are always considerable tubular and glomerular changes.
The actual amount of proteid matter found in the urine varies widely, being least in renal cirrhosis, where there is sometimes but a trace; on the other hand in some forms of chronic Bright's and in certain forms of lardaceous disease of the kidney, the amount of albumin is large and may reach forty grammes a day.

In acute Bright's, although the percentage of proteid matter in the urine is high, the amount lost is not very great owing to the small amount of urine secreted.

Tuberculosis of the kidney (Renal Phthisis)

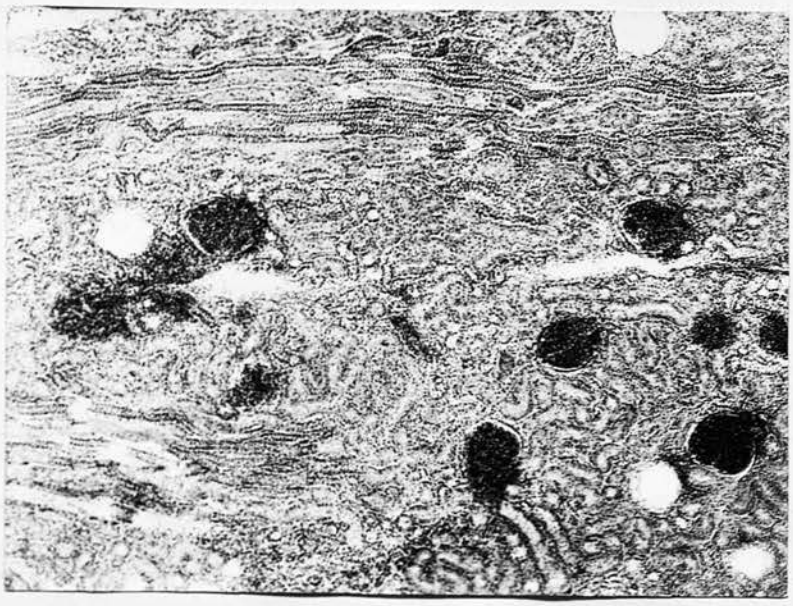
This condition is usually associated with tuberculosis elsewhere; thus there is commonly tuberculosis of the ureter, bladder, vesciculae seminales, vas-deferens, and testicle. Weigert, holds it is most probable that in most cases, the disease originates in the testicle and travels to other parts. This view is supported by the fact that the disease is rare in females. Renal Phthisis is often associated with pulmonary phthisis, which is probably in most cases the primary affection. In advanced cases the kidney is converted internally into a sack with irregular ulcerated walls, and divided partly into compartments by the remains of the septa, and the walls present adhering caseous matter. The capsule of the kidney is adherent. In an early stage, we find it may be, the apex of a pyramid, the seat of a small ulcer, with yellowish caseous walls, and with grey tubercles at the perriphery, in the substance of the pyramid.

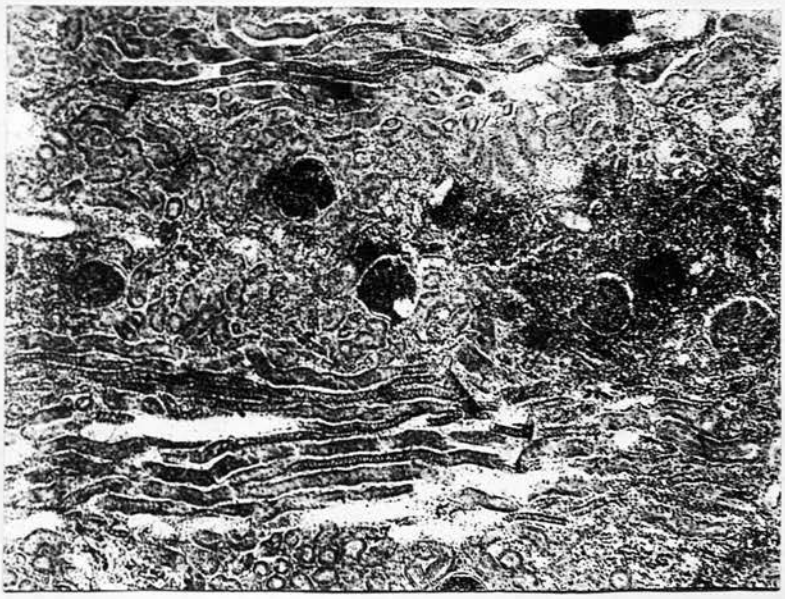
Primary cancer of the kidney.

This tumor is usually unilateral and is in most cases virtually a cancerous degeneration of the organ. The kidney may be completely converted into a tumor, which sometimes attains a very large size, but in some cases only a part of the kidney is involved, and in that case, while the affected part retains the general shape of the organ, although enlarged, the remaining piece of kidney has quite its normal appearances. To the naked eye it is as if a portion of kidney were transformed, and with a microscope itcan be seen, at the margin of normal and pathological, that the tumor is advancing by a conversion of the proper kidney tissue. The epithelium of the tubules is multiplying so as to form the cancerous epithelium, and is becoming irregular in form, while the cancerous stroma is being formed by the connective tissue of the organ. In regard to the form of cancer there are some cases in which the structure has been that of cylinder celled epithelioma; in others more that of ordinary cancer. A partial colloid transformation has been observed in a few cases.

Pyaemia of the Kidney, Is produced by pieces of fibrine and other material being carried in the circulation, producing emboli. For the most part the emboli are small, and are carried to the ascending arteries or the glomeruli before they are caught; hence the abscesses are mostly in the cortical substance, and are elongated in the direction of the arteries. They are frequently present in considerable numbers in both kidneys.

Microscopically - it is seen that the abscesses arise from obstruction of the arteries. Where the embolus has been recent the wall of the vessel, and the tissue in the immediate neighborhood of the embolus present evidence of necrosis, while around, there are multitudes of leucocytes occupying the interstitial tissue. In the embolus there are colonies of microbes in the midst of remains of the transported fibrine. When the abscess has fully formed these characters may be lost in the great multiplication of leucocytes. Besides in the arteries, colonies of microbes are to be found in the vessels of the glomeruli and also in the capillaries.



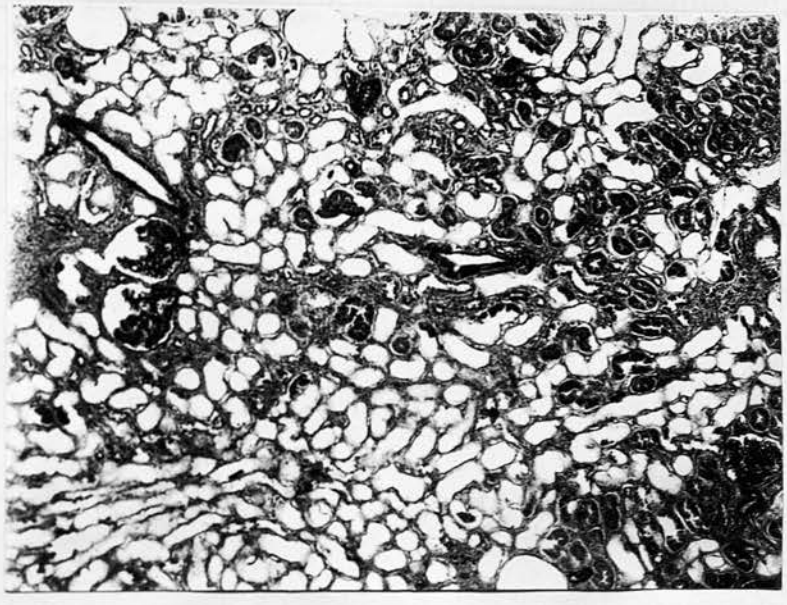


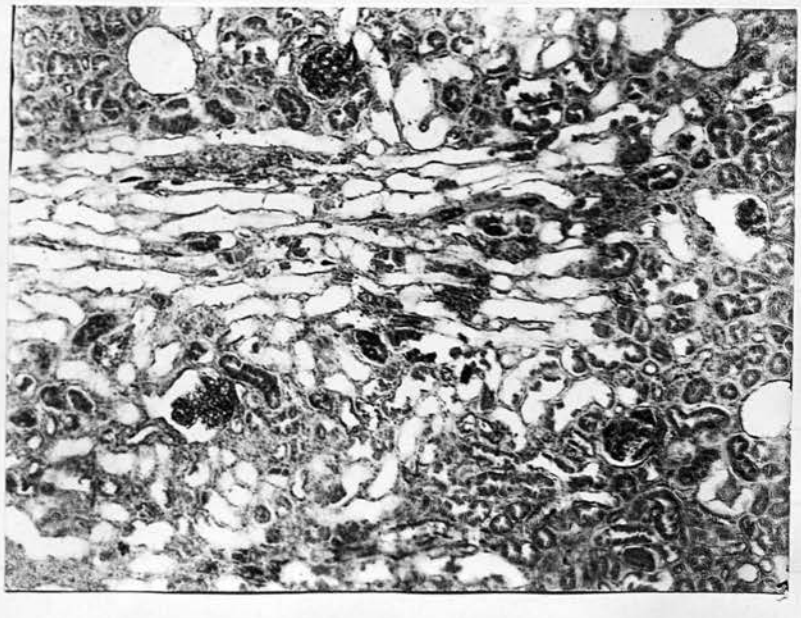
ACUTE NEPHRITIS. X 40.

GLOMERULAR AND TUBULAR CHANGES.

EXUDATION OF LEUCOCYTES.

TUBE CASTS.

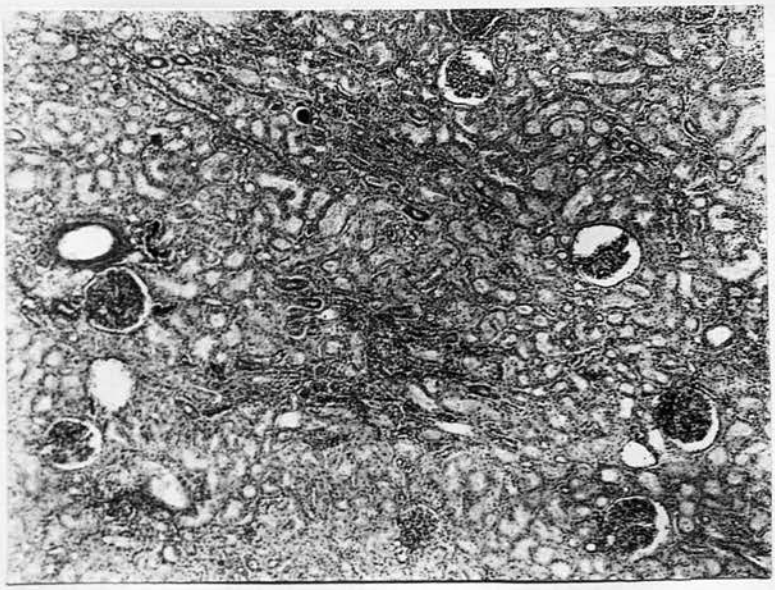


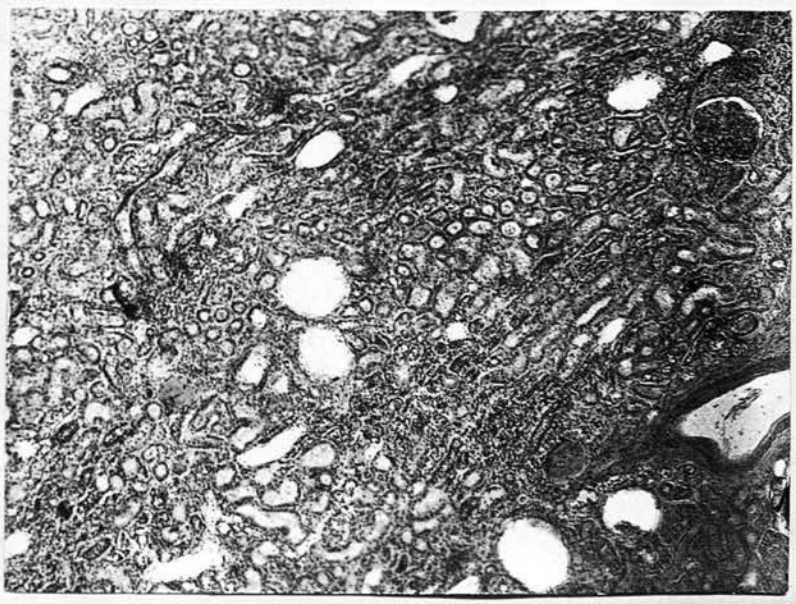


CHRONIC NEPHRITIS: X40.

GLOMERULAR DEGENERATION.

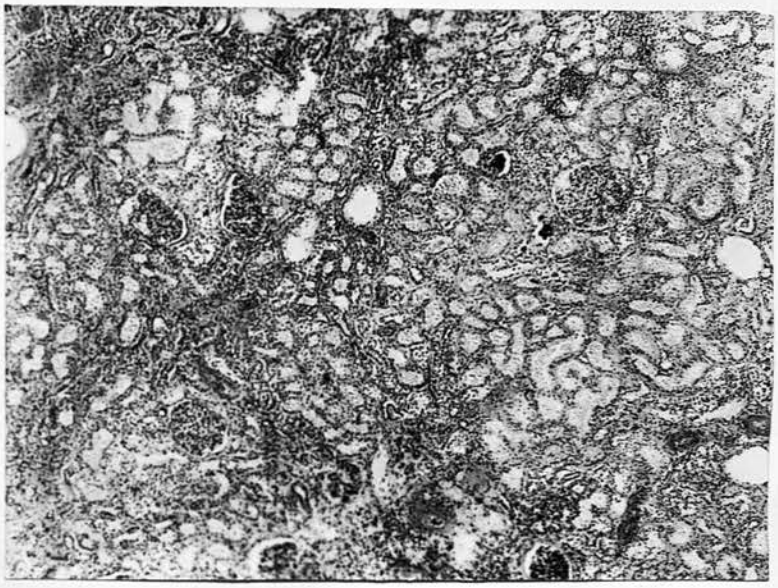
EPITHELIAL CHANGES.

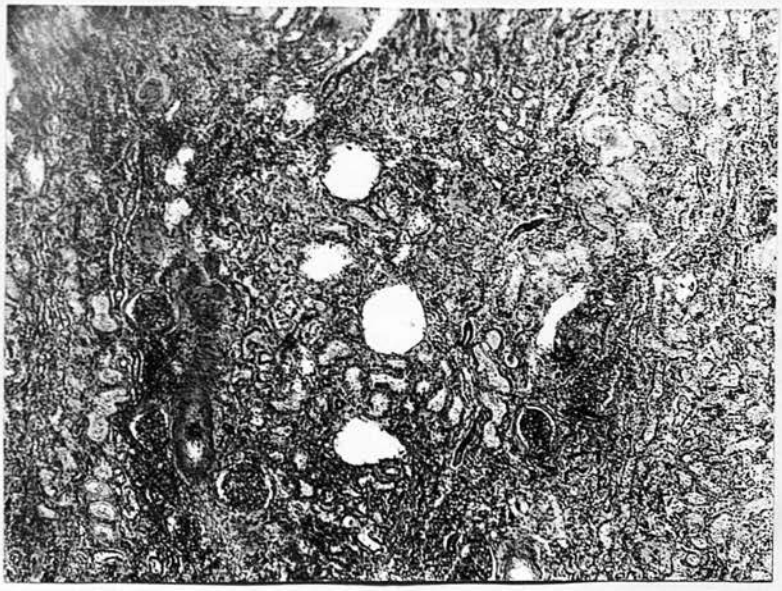




CIRRHOTIC KIDNEY :- X 40.

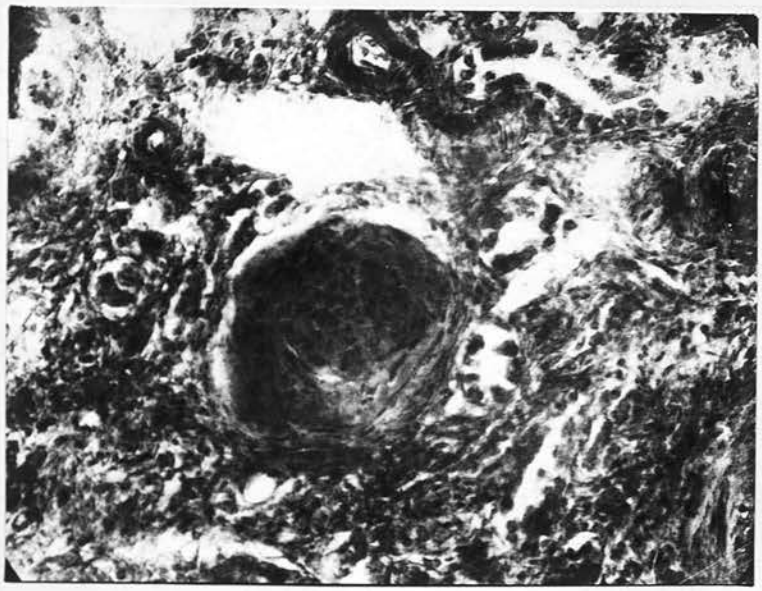
EARLY. CHANGES

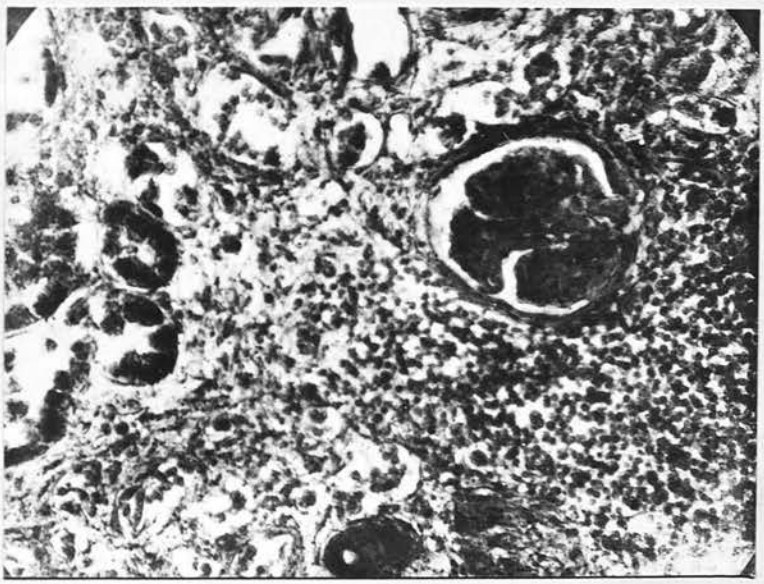




CIRRHOTIC KIDNEY :- X 40.

LATER STAGE.



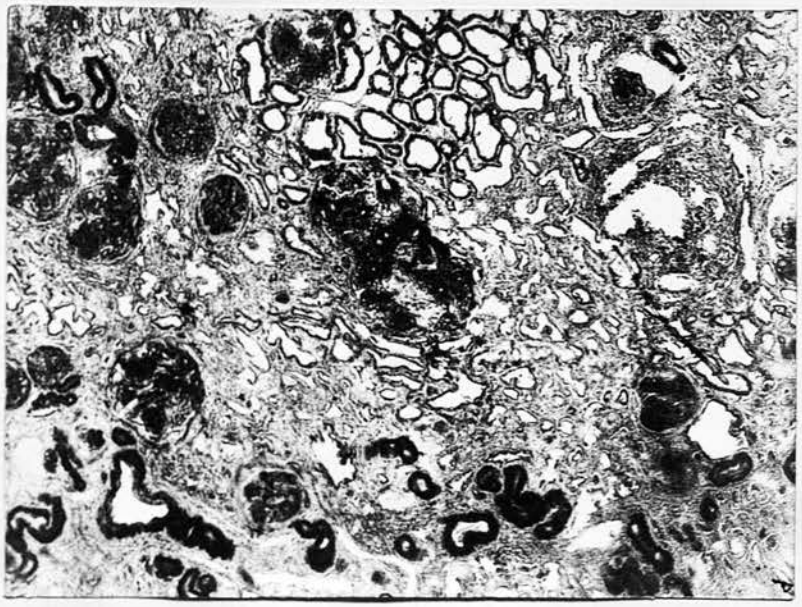


CIRRHOTIC KIDNEY:- X200.

PERICAPSULITIS.

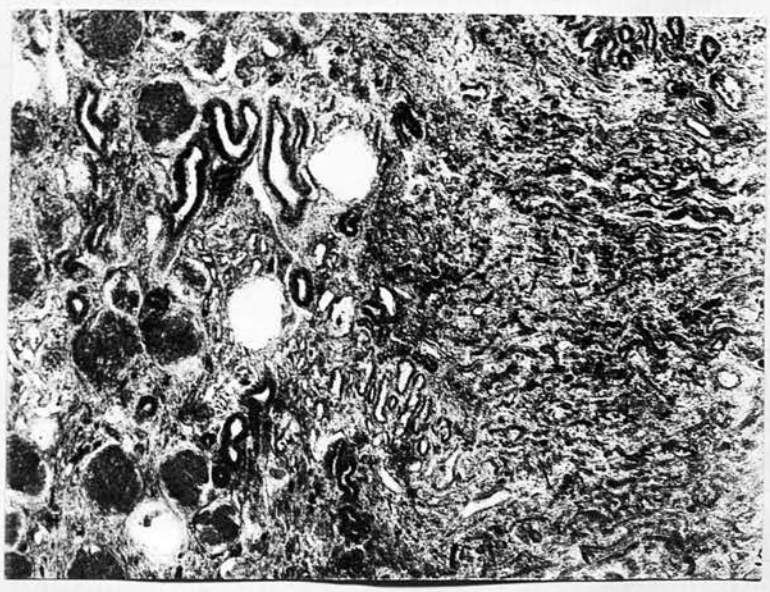
FIBROID CHANGE IN GLOMERULI.

ADVANCED CIRRHOSIS WITH WAXY DEGENERATION OF KIDNEY. X80.



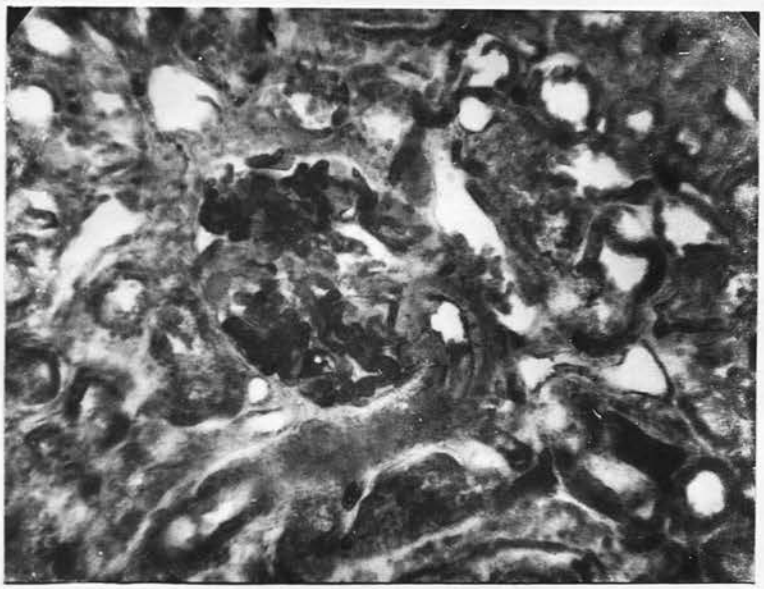
WAXY DEGENERATION:- X 40.

EARLY STAGE.



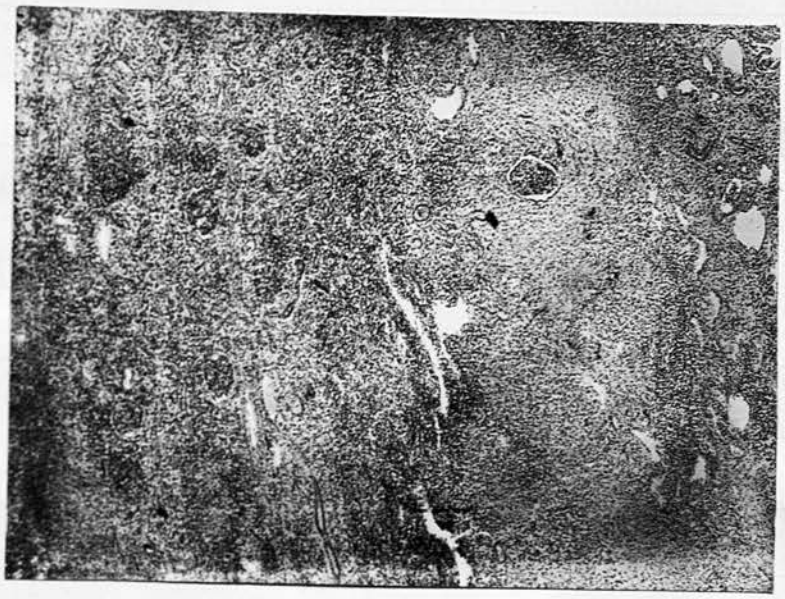
WAXY DEGENERATION. X 40

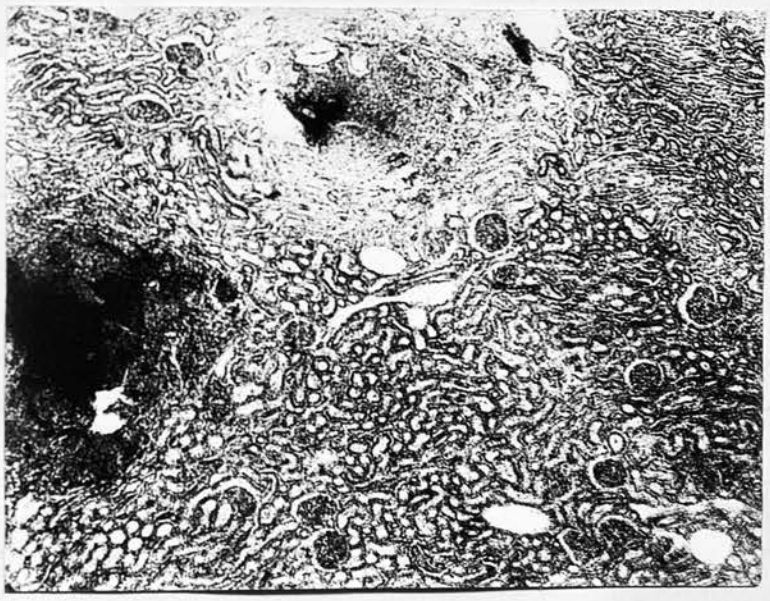
ADVANCED.



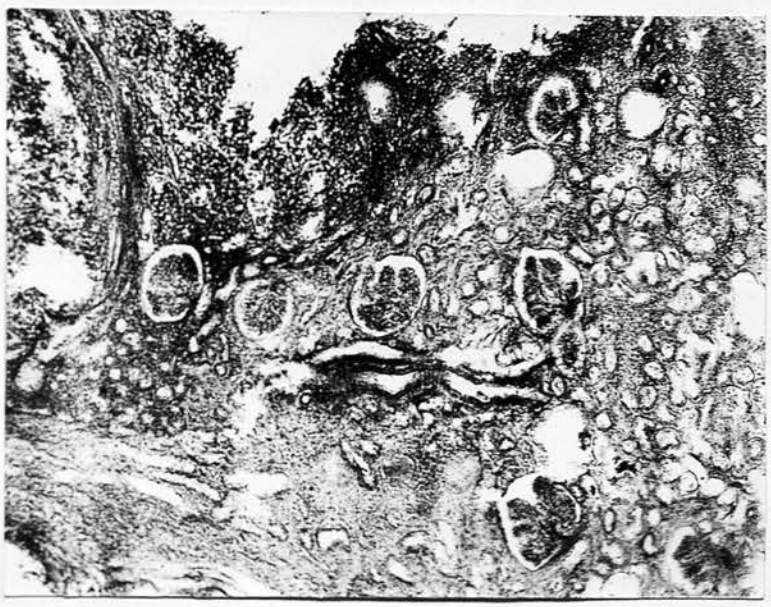
WAXY DEGENERATION X200.

GLOMERULUS. VESSELS INJECTED.





TUBERCULOSIS OF KIDNEY. X 40.



CARCINOMA OF KIDNEY. × 40.